PB83-213942

Pulmonary Function in Normal Humans with Exercise and Temperature-Humidity Stress

(U.S.) Health Effects Research Lab.
Research Triangle Park, NC

1982

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1. REPORT NO. EPA-600/J-82-090	2.	3. R PB83-213942
4. TITLE AND SUBTITLE Pulmonary function in normal humans with exercise and temperature-humidity stress (Journal Version)		5. REPORT DATE 1982 6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S) Ralph Stacy, Elston Seal, James Green and Dennis House		8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS Clinical Research Branch, Inhalation Toxicology Health Effects Research Laboratory US Environmental Protection Agency Research Triangle Park, NC 27711		10. PROGRAM ELEMENT NO. 11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Office of Research & Development Health Effects Research Laboratory US Environmental Protection Agency Research Triangle Park, NC 27711		13. TYPE OF REPORT AND PERIOD COVERED 14. SPONSORING AGENCY CODE EPA-600/11

15. SUPPLEMENTARY NOTES

Published In: J. Appl. Physiol., 53(4):1015-1018, 1982

16. ABSTRACT

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17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT Release to Public	19. SECURITY CLASS *(This Report)* Unclassified	21. NO. OF PAGES 5
	20. SECURITY CLASS *(This page)* Unclassified	22. PRICE

REPRODUCED BY NATIONAL TECHNICAL INFORMATION SERVICE U.S. DEPARTMENT OF COMMERCE SPRINGFIELD, VA. 22161

EPA-600/J-82-090
JOURNAL ARTICLE

Pulmonary function in normal humans with exercise and temperature-humidity stress

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STACY, RALPH W., ELSTON SEAL, JR., JAMES GREEN, AND DENNIS HOUSE. *Pulmonary function in normal humans with exercise and temperature-humidity stress.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 53(4): 1015–1018, 1982.— Fifty-eight normal young male human subjects were exposed for 4 h to comfortable conditions (22°C, 40% rh) or to heat stress conditions (30°C, 60% rh) with or without exercise. Exercise amounted to two 15-min sessions of treadmill walking at 6.7 km·h^{-1} (4 mph) with a 10% grade beginning at 105 and 225 min after entry into the chamber. Measurements of 15 pulmonary function variables were made *1*) before entry into the chamber, *2*) 5 min after the first exercise period, *3*) 5 min after the second exercise period, and *4*) 24 h after the end of the exposure period. The exercise, estimated to use about two-thirds of the subjects' maximum oxygen intake, produced no statistically significant ($P < 0.01$) changes in pulmonary function parameters. Heat stress produced significant changes in forced vital capacity, and possibly significant interactions were observed in peak expiratory flow and forced expiratory flow at 25% of vital capacity. Effects of the two factors appeared to be additive. Changes with exercise and heat stress were associated with reversal of a progressive decrease of airway resistance seen in subjects at rest in a comfortable environment.

male subjects; heat stress; forced vital capacity; peak expiratory flow; forced expiratory flow; comfortable environment

IN SEVERAL FIELDS of research (e.g., air pollution effects) it is recognized that pulmonary function can be influenced by the imposition of exercise and thermal stresses. With but a few exceptions (1, 4–6, 8) research on pulmonary function with these added stresses has assumed that there is no interaction of these stresses with other experimental variables. The experiments described in this paper were designed to provide basic information for two specific stresses, exercise and temperature-humidity.

The data presented herein represent a well-defined and validated normal-subject clean-air data base. It is expected that from these data, information can be extracted on the effects of exercise and high temperature and humidity on pulmonary function measurements. Also, these data can help to identify interactions between measurement conditions that should be taken into account in studies done under such conditions.

METHODS AND PROCEDURES

This experiment consisted of studies before, during, and after a 4-h exposure of the subjects in a controlled environment chamber.

For the variable exercise, the experimental conditions were *1*) at rest and *2*) two exercise sessions. One of the exercise sessions was performed at about 2 h after entry into the chamber and the other session at about 4 h after entry. Each session consisted of walking on a treadmill at 6.7 km·h^{-1} (4 mph) at a 10% grade. From previous experience, it was estimated that this work level utilized about two-thirds the maximum oxygen intake of the subjects used.

The levels of temperature and humidity used in these studies were *1*) a "comfortable" level (22°C, 40% rh) and *2*) a "heat stress" level (30°C, 60% rh.)

Subject selection. Fifty-eight subjects were studied in four separate groups, with 14 in the low temperature-humidity (T/H)-rest group, 16 in the low T/H-exercise group, 13 in the high T/H-rest group, and 15 in the high T/H-exercise group. The subjects were young (age 18–40) healthy males recruited from the student bodies of several local universities. Subjects were given a complete physical examination, including complete medical histories and screening blood chemistries. Each subject was given a Minnesota Multi-Phasic Inventory test, and each subject was given a skin test utilizing 16 antigens, 4 concentrations of methacholine, and saline. Smokers, subjects with signs of recent pulmonary infections, and those with positive skin tests or histories indicating allergies were not used.

Measurements. These studies were performed in the air pollution exposure chambers of the US Environmental Protection Agency Clinical Studies Facility in Chapel Hill, NC. These facilities have been described in detail elsewhere (12).

Pulmonary function measurements. A CPI model 3100 body plethysmograph interfaced to a DEC PDP 11/40 computer system was used for the acquisition and analysis of data from which functional residual capacity (FRC), thoracic gas volume (TGV), airway resistance (Raw), and specific airway conductance (sGaw) were derived.

A CPI model 220 rolling-seal spirometer, also inter-

faced with the PDP 11/40 computer, was used to perform three standard maneuvers: *1*) forced expiratory maneuver, *2*) normal breathing and standard vital capacity maneuver, and *3*) forced inspiratory maneuver. Analyses of data from these maneuvers produced values for 11 variables reported herein. These were forced vital capacity (FVC), forced expiratory volume at 1 s (FEV_1), FEV_1/FVC, peak expiratory flow (PEF), forced expiratory flow at 25% of VC (FEF_{25}), tidal volume (V_T), frequency (f), VC, forced inspiratory VC (FIVC), forced inspiratory volume at 1 s (FIV_1), and peak inspiratory flow (PIF).

To eliminate the effects of deep-breathing maneuvers on airway resistance, plethysmography was always performed before spirometry. Pulmonary function measurements following exercise were performed after at least 5 min of recovery from the exericse.

Exercise testing. Exercise sessions were 15 min of walking on a treadmill at 6.7 $km \cdot h^{-1}$ (4 mph) and 10% elevation. The total period in the environmental chamber was 4 h, with exercise at midperiod (beginning at 105 min after chamber entry) and at the end of the exposure (beginning at 225 min after chamber entry). Physiological measurements were made *1*) before exercise, with the subject standing at the treadmill and ready to start the exercise, *2*) 5 min into the exercise, *3*) 10 min into the exercise, and *4*) during the last minute of the exercise.

The measurements made included heart rate, respiratory rate, V_T, ventilation volume, oxygen consumption, and carbon dioxide production. Airflow was measured with a Fleisch pneumotachometer mounted on the frame of the treadmill; gas analysis was performed with a Perkin-Elmer model MGA-1000 mass spectrometer. Heart rate was calculated from single-lead electrocardiographic signals, and heart rate calculation was continued through 3 min of recovery following exercise.

Statistical analysis. For analysis, the mean of the two best trials of any maneuver was used. Multivariate analysis of variance was used to test three hypotheses for each variable. These were *1*) no difference in response levels at the two T/H levels, *2*) no difference in response levels at rest and with exercise, and *3*) no interaction between T/H and exercise stress. These analyses were performed on the patterns of response, represented by values obtained at all four conditions of measurement.

RESULTS

Table 1 presents the demographic data means for the four groups of subjects included in this study. Examination of these data indicate that there are small differences in age and size between these groups, but these are not large enough to account for differences seen in the experimental data.

The probabilities computed for the three hypotheses tested are listed for the 15 variables considered physiologically important in Table 2. Interpretation of these *P* values depends on the degree of conservatism the observer wishes to use. We have chosen to consider that in this experiment, a moderately conservative critical *P* value is 0.01. The calculated *P* values given in Table 2 are for differences in total patterns (including values for 0, 2, 4, and 24 h), and the statistics used for analysis of such differences of patterns are inherently conservative.

TABLE 1. *Comparison of age, size, and vital capacity of subjects in the four groups studied*

Cohort	*n*	Mean Age, yr	Mean Ht, cm	Mean Wt, kg	Vital Capacity, ml
Low T/H with no exercise	14	23.8 ± 0.4	180 ± 4.4	74.6 ± 8.7	5,449 ± 130
Low T/H with exercise	16	22.8 ± 3.8	182 ± 6.0	74.4 ± 9.4	5,580 ± 132
High T/H with no exercise	13	25.2 ± 6.0	182 ± 4.8	78.4 ± 8.7	5,552 ± 226
High T/H with exercise	15	22.6 ± 2.4	182 ± 5.6	72.1 ± 7.4	5,527 ± 181

Values are means ± SE. T/H, temperature/humidity.

TABLE 2. *Probabilities associated with tests of hypotheses using exact multivariate F test*

Parameter	H1, No T/H Effect	H2, No Exercise Effect	H3, No T/H Exercise Interaction
FVC	0.009*	0.635	0.193
FEV_1	0.780	0.912	0.098
FEV_1/FVC	0.322	0.798	0.205
PEF	0.699	0.116	0.045
FEF_{25}	0.248	0.518	0.014
FRC	0.966	0.802	0.140
Raw	0.091	0.093	0.934
TGV	0.647	0.578	0.675
sGaw	0.015	0.041	0.738
V_T	0.714	0.030	0.559
f	0.380	0.281	0.537
VC	0.666	0.228	0.201
FIVC	0.118	0.183	0.986
FIV_1	0.261	0.307	0.504
PIF	0.633	0.540	0.540

Degrees of freedom for all tests were 3 and 52. H, hypothesis; see text for definition of abbreviations. *$P < 0.01$.

Thus the effect of temperature and humidity on FVC is the only effect that can be considered clearly significant. Other changes that suggest significance are likely due to chance. The patterns of change of other variables resembled those of FVC.

The nature of the effect of T/H on FVC can be seen in Fig. 1. This graph presents the curves for low and high T/H, when data taken at rest and with exercise have been pooled for each T/H condition. In the low T/H groups, the end exposure (*condition 3*) and 24-h postexposure (*condition 4*) means returned to approximately base-line level. However, in the high T/H groups the FVC did not return to base line but remained elevated. The peak difference of FVC (1.39%) is a small difference compared with other differences noted in this study.

Analysis of the data produced during exercise showed that high T/H conditions did not significantly alter responses to exercise. Analysis of the bronchial challenge curves indicated that neither exercise nor high temperature and humidity significantly altered the position or slope of the bronchoconstrictor response curve. Finally, no correlation between skin test responses and pulmonary function responses was observed.

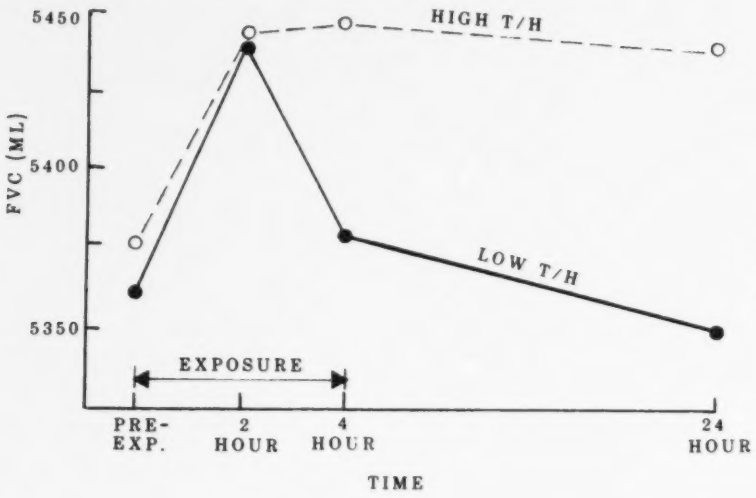

FIG. 1. Effect of heat stress on forced vital capacity (FVC), with data from rest and exercise groups at each heat stress level pooled. T/H, temperature and humidity.

DISCUSSION

In this research, the four experimental groups consisted of different subjects (they did not serve as their own controls), and statistical methods used were selected with this in mind. This one-time use of subjects made subject recruitment much easier. The experiment was carefully designed to take this factor into account.

Of the variables measured in this study, only FVC was significantly altered by warming and humidifying the air breathed.

The "resting" V_T values were higher than those usually reported. Actually, V_T was measured while the subjects were standing and were involved in a spirometric measurement regime. Combined with the anticipation of the maneuver to be performed, these conditions can account for the high values noted.

An important observation is that after 2 h of rest in a low T/H environment, the sGaw increased by 21.7% and by the end of 4 h, by 26.1%. Twenty-four hours later (after normal activity) the sGaw had returned nearly to base line (+8.7%). Thus simply resting in a comfortable environment results in a relaxation of the airways and a decrease of Raw. Either exercise or warming and humidifying the air breathed eliminates this change.

What appeared to be relatively large differences in the 24 h postexposure values for sGaw and PEF must be attributed to greater variability of the 24-h data. This variability is associated with the fact that subject activities were not controlled in the postexposure period, and those activities surely varied between subjects.

It is well known (2, 9, 11) that exercise brings about large increases of Raw and decreases of sGaw in asthmatics; there is a large volume of literature on what is known as "exercise-induced asthma." However, there is far less information on effects of exercise per se on the pulmonary function of young healthy subjects. In this study, we have seen that after the first exercise session (2 h into the exp), there was no change of sGaw from the base line measurement. Thus it is apparent that in normal subjects, the hyperventilation produced by exercise simply prevents the airway relaxation that occurs at rest.

It has been shown (3, 10, 11, 12) that exercise-induced bronchoconstriction is prevented by having the subject breathe warmed humidified air. This has been associated with the effect of cooling by inhalation of cool dry air. From our studies, it appears that in normal healthy subjects both exercise and high T/H prevent the decrease in Raw that occurs at rest in comfortable conditions.

The time constant of recovery of pulmonary function after exercise appears to be 5–10 min (3, 7–9). Thus the scientist measuring pulmonary function more than 5 min but less than 20–30 min after exercise must take into account the fact that the measurement is being made during a transient period of recovery. Our measurements were made about 10 min after exercise; the differences noted are probably smaller than they would have been had it been possible to make them immediately after the exercise.

The authors express their appreciation to Dr. Keith Muller for his early contribution to the experimental design, to Dr. William McDonnell, and nurses Martha Guttu and Jean Litton for their work in connection with subject selection and processing, and to Dr. Jack Roger, Louis Raggio, and Keith Edleman of Rockwell International for their implementation of this research.

This paper has been reviewed by the Health Effects Research Laboratory, US Environmental Protection Agency and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendations for use.

Received 7 December 1981; accepted in final form 18 May 1982.

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